VOL. XVI, No. 2 MARCH-APRIL, 1955

UNIVERSITY OF MICHIGAN
MAY 9 1955
MEDICAL
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BORDEN'S REVIEW

of

NUTRITION RESEARCH

NUTRITION AND ENDOCRINE FUNCTION

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NUTRITION RESEARCH

Reviews and summaries of research information concerning nutrition and foods. Published bi-monthly, by THE BORDEN COMPANY; officers of the company: Theodore G. Montague, president; E. L. Noetzel, treasurer; D. T. Orton, secretary. Editorial office: 350 Madison Avenue, New York 17, N. Y.

NUTRITION AND ENDOCRINE FUNCTION

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The nutritional state of an individual means the condition of all tissues of his body, both as to structure and function, as influenced by the food he eats, the amount of food constituents absorbed, and the efficiency of utilization of those constituents. Deficiencies of some food constituents have marked effects on tissue structure; in other cases little evidence of structural changes can be found but alterations in function are manifest. Alterations in nutrition may not produce changes in all tissues, or at least with equal rapidity, but deviations in structure and in function are likely to be more widespread than would be judged from reports of research.

Enthusiasm for one's own work is understandable; in biological research this enthusiasm sometimes may cause confusion and delay progress by restricting attention to one particular aspect of a general problem. In this review we shall note several instances in which the focusing of attention upon a single observation has limited, for a time, knowledge of wide-spread changes produced by an alteration in nutrition.

The endocrine glands are highly active, specialized tissues which, by producing specific hormones, control the utilization of food constituents. The hormone produced in the thyroid enters the blood stream and is circulated throughout the body; by processes not yet known it controls the production of energy. Insulin, the hormone of the pancreas, regulates the utilization of carbohydrate. Back of these and other endocrine glands is the general superintendent, the pituitary, the hormones of which control the production of hormones by all other endocrine organs. The activity of the thyroid, and the production of the thyroid hormone, are controlled by the thyrotropic hormone sent from the supervising pituitary gland. While these endocrines have regulatory functions and do have a real effect on the utilization of food constituents, it would be expected that they would be influenced by nutrition. The close interrelation between endocrine function influencing the utilization of food constituents and consequently the nutritional state of tissues, and the reverse effect of nutrition upon activities of the endocrine glands has often been overlooked. L. T. Samuels (1) has stated clearly this failure:

"The student of nutrition has rarely taken cognizance of the fact that the animal on which he experiments is a carefully balanced dynamic

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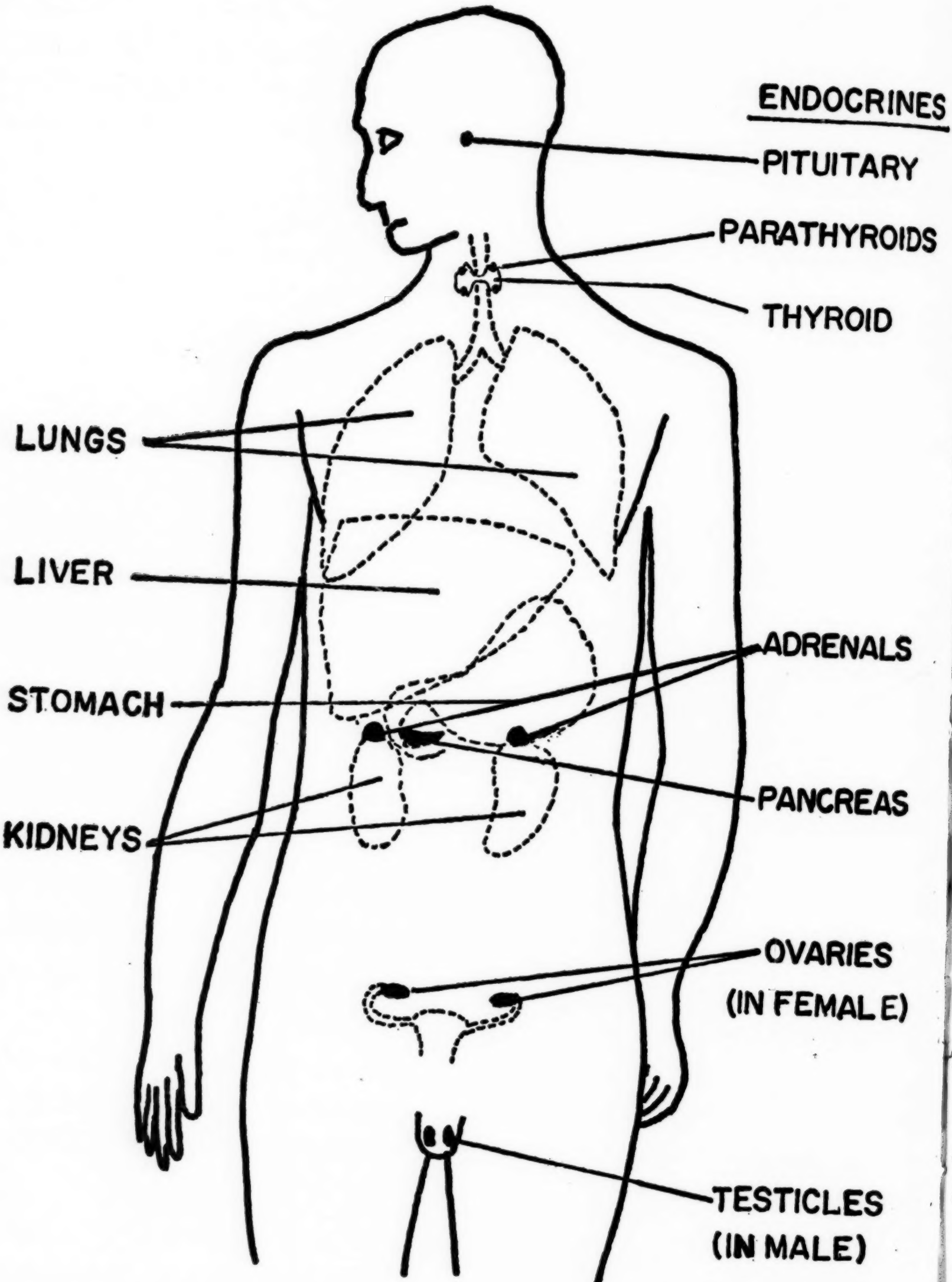

LOCATION OF ENDOCRINE GLANDS

system controlled in many ways by internal secretions. The fact that the influence of the endocrine glands is often offset by changes in appetite has also tended to obscure their role when the animals have been fed *ad libitum* . . . Conversely, it is seldom that the endocrinologist has emphasized the nutritive significance of his observations although, when one stops to look upon them, one realizes their importance."

In this review recent evidence regarding the dual and converse effects of the endocrines upon nutritional state and of nutrition upon the endocrines will be discussed. No claim is made for completeness. Topics have been selected because they seemed to be of interest. Omission of topics or of particular pieces of work has been made necessary by space limitation.

Inanition and endocrines

The total amount of food eaten by an individual is determined by appetite and hunger. Animals and humans eat because they are hungry and generally stop eating when hunger has been satisfied and a feeling of well-being has been attained. A feeling of emptiness with a consequent craving for food causes animals to eat. Humans, in civilized conditions, frequently eat because of custom. The selection of particular foods to satisfy hunger may be determined by "appetite," using that word in the popular, loose way meaning pleasant sensation, or by a process of selection based on psychological and physiological processes of which we know very little. To cite one example: Richter (2) showed that rats select varying proportions of protein, fat and carbohydrate when supplied with different B vitamins, presumably adjusting the intake to actual need. Conversely, lack of a single vitamin or indeed of any nutrient causes a reduction of appetite or hunger, so that food consumption is lessened. This restriction in food intake produces in the animal a state of inanition or undernutrition which could be brought about also by giving to an animal a complete diet in a consistently smaller amount than hunger would cause it to eat if food were freely available. The effects of continued maintenance on restricted food intakes (inanition) and of complete starvation have been studied extensively on experimental animals, and, in a great deal of nutritional research, have been ignored. Chronic inanition of various degrees up to complete starvation has occurred repeatedly in human history in famines. Inanition was brutally and willfully induced in some prisoner-or-war and concentration camps in World War II. Because of the great practical importance the effects of sustained inanition have been purposely studied on human subjects in carefully designed experiments.

A sustained restriction of food intake, sufficient to cause marked loss of weight, has been shown to affect the endocrine glands of animals and of

humans. From a number of reports dealing with observations made during World War II, two can be used as examples. Sydenham (3) described conditions in a Japanese-controlled camp for civilians at Hong Kong. In a group of 655 women 54 percent showed cessation of menstruation. In many cases this began before food restriction and may have been initiated by emotional shock, but the cessation of the sexual rhythm persisted throughout a long period during which food intake was reduced; sexual rhythm was restored soon after additional food, especially meat, became available. Jacobs (4), who was with U. S. forces in Japanese camps for 38 months, reported comparable interference with sexual processes in men. Jacobs stated that prolonged inanition produced symptoms similiar to those resulting from surgical removal of the sex glands. Return to adequate food supplies restored sexual function and there was no permanent impotence or sterility.

The effects of sustained inanition were thoroughly studied by Keys and associates (5) in a group of 32 volunteer subjects, all men. The observations were summarized by the authors: "The results of semi-starvation in man in many ways bears a resemblance to the clinical symptoms of endocrine gland deficiency diseases. A low pulse rate, low blood pressure, and low basal metabolic rate are characteristic of both semi-starvation and hypothyroidism. The emaciation of semi-starvation is, superficially at least, suggestive of Simmond's disease. The pigmentation of the skin in the semi-starved individual has some resemblance to the pigmentation of Addison's disease. The loss of the sex-drive in males, cessation of menstruation in females, decreased incidence of hyperthyroidism and diabetes mellitus, and increased incidence of myxedema among the victims of severe famine are additional indications that endocrine gland function is disturbed by semi-starvation."

Zubiran and associates (6) made careful observations on a large group of Mexicans who had suffered from chronic malnutrition. In both men and women there was evidence of impairment of the sex glands and diminished output by the pituitary of the hormone which is normally sent from that gland to stimulate the sex glands. The adrenal glands were diminished in size and function and, again, there was evidence that this was due to less stimulating hormone from the pituitary. Similar observations on the thyroid and other glands provided a picture of inanition decreasing the activity of the pituitary with consequent impairment of the subsidiary endocrines. However, it has been pointed out by several investigators, particularly Samuels (7), that the various stimulating hormones put out from the pituitary are not lessened in equal amounts simultaneously. The chemical messenger from the pituitary to the sex glands is most sensitive to inanition and impairment of sex function is likely to be first evidence of the effects of inanition on the endocrines.

Prior to these observations on humans the effects of semi-starvation had been examined in experimental animals by Mulinos and Pomerantz (8). One of the great advantages of animal experimentation was evident; it was possible to remove the various endocrines and minutely examine changes in structure. Consequently, Mulinos and Pomerantz were able to present in detail the picture which was later described from the human observations. The experimental animal was altered by inanition in ways similar to those caused by surgical removal of the pituitary with consequent absence of the hormones sent from the pituitary to activate the subsidiary sex glands, thyroid, adrenals and other endocrines. Marked reduction in food intake for a sustained period has been shown in both animals and humans to depress the stimulating effects of the pituitary on the various endocrines and these, in turn, are lessened in activity.

Cursory consideration of these effects of chronic undernutrition would indicate that the observations are of importance in the practical application of nutrition to humans. This is, of course, true but there is another aspect, perhaps not clear at first sight. Numerous investigators have been, and are, concerned with studies on single nutrient deficiencies. One of the early effects of the dietary lack of a nutrient is lessening of appetite with a consequent decrease in food consumption. There is produced, not only a deficiency of the studied nutrient, but also a state of chronic inanition. How many of the changes in the experimental animal are due to the planned deficiency of the nutrient under study, and how many result from the unplanned, but very real, inanition? In the light of the above discussion, how many of the changes can be attributed to the depressing action of inanition on the pituitary and the dependant endocrine glands? These questions have been overlooked frequently in the interpretation of research findings; they are of sufficient importance to warrant mention several times later in this review.

Effects of dietary protein, fat and carbohydrate on endocrines

While the effects of general undernutrition on the endocrine glands have been studied, it is not entirely clear whether the results should be attributed to an energy deficit or to the lack of a particular type of nutrient. Since the pituitary is impaired first and since its messenger hormones are proteins which must be synthesized from amino acids, it is reasonable to consider whether a lack of protein may not be the primary factor. The clinical report of Sydenham (3) stressed that normal sexual rhythm was restored to the women prisoners at Hong Kong when a supply of animal protein, as meat, became available. It cannot be concluded that high quality protein was itself responsible for recovery since the meat undoubtedly contained appreciable

fat and provided a significant increase in calories. However, Samuels (7) has placed emphasis on protein, stating that a protein deficit, even though small, would cause irregularities in sexual function, if the deficit were sustained for some time. Further studies are needed to determine the role of protein and of individual amino acids in the effects of inanition.

Panos and Finerty (9) have described the effects of dietary deprivation of fat on the endocrine glands of both male and female rats. Changes in the sex glands, thyroid and adrenals suggested that the activating action of the pituitary was depressed.

In a series of reports Best and Haist, and later Haist and associates (10, 11, 12) provided interesting and clinically important evidence about the effects of dietary fat and carbohydrate on the pancreas and its content of insulin. High-fat diets caused a marked decrease in the production of insulin. Unlike the effects of inanition, exercised primarily through the pituitary, this effect of dietary fat is directly on the pancreas. These findings raise several practical and important questions. Most people in the United States and in Canada voluntarily choose generous proportions of fat in meals. Does sustained use of high-fat meals depress the pancreas and lead to an increased incidence of diabetes? In many diabetics there is still some insulin produced by the pancreas. Should the pancreas of a diabetic patient be "rested," or at least depressed in function, by the use of a high-fat diet, or should the pancreas be stimulated to produce as much insulin as possible by the use of a high-carbohydrate diet? Clear answers to these questions are not available.

Mineral deficiencies and endocrines

The relation of iodine intake to thyroid function and to the production of simple goitre is a familiar one. It is the only accepted link between soil composition and human disease. In North America and elsewhere the simplest and surest way to ensure that a population will obtain sufficient iodine without harmful effects is the general use of iodized salt. It is a matter of gratification to the writer that Canada has made compulsory the iodization of all table salt. Opponents of the iodization of salt used by humans have cited two types of objections: (a) possible harm from too much iodine, and (b) apparent exceptions to the iodine-deficiency explanation of endemic goitre. In a recent address Marine (13), to whose life-long work on iodine metabolism we are so greatly indebted, has discussed the objections and has stated that the main obstacles to goitre prevention are economic, political, and social factors. In Canada the main obstacle over some years was the inertia of the medical profession, and more particularly, of public health officials.

It may well be that supplies of other minerals are essential for normal functions of endocrine glands. Zinc is present in high concentration in the pancreas and may be linked to the controlling action of insulin on carbohydrate utilization.

Ascorbic acid and adrenals

Ascorbic acid, not then identified as vitamin C, was isolated from adrenal glands by Szent-Gyorgyi (14) in 1928. In 1934 Bessey, Menten and King (15) examined the various tissues of guinea pigs which had been rendered deficient in ascorbic acid and reported that the most characteristic change was in the cortex of the adrenal gland with a marked loss in fat and cholesterol. Ten years later an important observation was made by Long and coworkers (16). By that time there was available a fairly pure preparation of the hormone produced by the pituitary to stimulate the activity of the adrenal cortex, a hormone popularly known as ACTH. A single dose of this hormone caused a very prompt disappearance of one-third of the large amount of the vitamin from the adrenals; considerable cholesterol was lost, also, but more slowly than was the ascorbic acid. The main effect of ACTH is to stimulate the adrenal cortex to produce hormones which are chemically related to cholesterol; Long and his associates concluded that the adrenal hormones were made from cholesterol and that the vitamin, ascorbic acid, was necessary to bring this about. This attractive theory has caused a great deal of research in a number of laboratories; unfortunately not all of the results have supported the theory and a conflict of opinion has developed.

It has been pointed out that a severe deficiency of ascorbic acid should prevent the production of adrenal cortical hormones. For such a study the rat, the species used by Long, cannot be employed because the rat manufactures sufficient ascorbic acid to meet requirements and a deficiency cannot be produced by dietary lack of the vitamin. Guinea pigs do need ascorbic acid in the diet and will develop the deficiency disease, scurvy, if deprived of the vitamin. Hyman and associates (17) produced scurvy in guinea pigs and found that the hemorrhages, characteristic of the disease, could be lessened and life prolonged by administering either the pituitary hormone ACTH or the adrenal hormone cortisone. Since ACTH was effective it was concluded that this hormone could exert its usual stimulating effect to cause a production of adrenal hormone even if there was a severe deficiency of ascorbic acid. After a careful study of tissue changes, Wolbach and Maddock (18) were unable to find any effect of cortisone on scurvy in guinea pigs. Indeed, they concluded that a deficiency of ascorbic acid had no peculiar effect on the adrenal glands. In this connection it should be recalled that Mulinos and

Pomerantz (8) described an increase in the size of adrenal glands and a decrease in the ascorbic acid content of those glands to be caused by inanition.

The conflicting claims regarding the effects of ascorbic acid deficiency on the adrenals and regarding the possible purpose served by the normally large amount of ascorbic acid in the adrenals has been reviewed very critically by Meiklejohn (19). He has pointed out that the concentration of ascorbic acid in the adrenals appears to be very great but that these glands probably do not contain even one percent of the total amount of the vitamin present in the entire body, and that there is also a high concentration of ascorbic acid in the pituitary. If ascorbic acid is essential for the production of adrenal hormones there should be a decreased output of the hormones in scurvy; no such evidence exists. Moreover, scurvy should cause the development of conditions similar to those seen in Addison's disease which is brought about by a lack of adrenal hormones. Such is not the case. If ascorbic acid is necessary for the formation of adrenal hormones from cholesterol, there should be an accumulation of cholesterol in the adrenal glands of ascorbic acid-deficient guinea pigs; actually there is a marked decrease in cholesterol.

The present reviewer is of the opinion that the original observations of Long and associates (16) have been substantiated frequently and that the pituitary hormone ACTH does cause a reduction in cholesterol and ascorbic acid in the adrenal glands. It is not clear that this two-fold effect is specific for ACTH nor is it clear that other tissues are not altered similarly. The interesting theory of ascorbic acid function is not established and it is becoming increasingly clear that the primary function of ascorbic acid has not yet been elucidated.

Pantothenic acid and the adrenals

In 1939 Morgan and Simms (20) reported that rats deprived of several B vitamins showed a decrease in the size of the adrenals. A somewhat similar finding was described by Daft and Sebrell (21) later in the same year. Within a short time attention was focussed on the B vitamin, pantothenic acid, as the specific factor and the theory was advanced that pantothenic acid is necessary for the normal activities of the adrenal gland. Surgical removal of the adrenal glands impairs the ability of an animal to get rid of excess water; Gaunt and associates (22) found that a similar inability to handle water was present in pantothenic acid-deficient rats and also in the late stages of riboflavin deficiency. The normal ability to dispose of water could be restored by supplying either the deficient vitamin or adrenal cortical hormones. Krehl and associates (23, 24, 25) have supplied substantial evi-

dence regarding the relation of pantothenic acid to the functions of the adrenal glands, evidence which is summarized briefly: The adrenal stimulating hormone, ACTH, had little effect in stimulating the production of adrenal hormones in pantothenic acid-deficient rats. In young rats a deficiency of pantothenic acid caused damage to the adrenals. This was intensified by administering ACTH, but was prevented by giving cortisone, an adrenal hormone. The vitamin deficiency caused a reduction in the amount of cholesterol in the adrenals, administration of ACTH further reduced the cholesterol, while cortisone prevented any change in adrenal cholesterol. It has been known since the work of Lipman and Kaplan (26) in 1947 that pantothenic acid is an active component of coenzyme A which makes possible the utilization of acetate for the formation of cholesterol and possibly adrenal hormones. Krehl and associates (25) suggested that the depletion of coenzyme A in the cortex of the adrenal gland, resulting from pantothenic acid deficiency, causes the functions of the adrenals to be impaired. The resultant decrease in adrenal hormones induces the pituitary to put out more ACTH; this cannot produce greater adrenal activity but does cause an increase in size of the gland with damage and hemorrhage. In agreement with this explanation, or at least with the data leading to it, are extensive observations by Ralli and Dumm (27).

Partial disagreement with the above observations has been expressed by Ershoff (28, 29). A lack of pantothenic acid, like other nutrient deficiencies, causes a loss of appetite and decreased food intake. The experimental animals suffer from the vitamin deficiency and from inanition. Damage to the adrenals may be due to either or both factors. Ershoff has concluded that both factors are responsible. Using four different tests of adrenal function Ershoff and associates decided that the adrenal glands of pantothenic acid-deficient rats were able to respond reasonably well to ACTH. The inanition explanation was examined, also, by Perry and co-workers (30) who concluded that pantothenic acid was definitely involved in adrenal function. A further demonstration of impairment of the adrenals by pantothenic acid deprivation has been supplied recently by Hurley and Mackenzie (31). There is now a great deal of evidence that a deficiency of pantothenic acid impairs the functions of the adrenal glands and the depletion of coenzyme A in the glands is the most likely explanation. Nevertheless, it would be unfortunate if the impression were given that the adrenal glands were the only site of activity of pantothenic acid (or of coenzyme A).

Other vitamins and the endocrines

In 1935 Coplan and Sampson (32) described an effect of vitamin A deficiency on the thyroid gland in rats, the effect being enlargement of the

gland in females and reduction in size in males. Kelley and Day (33) reported a somewhat reverse situation. They caused a depression in thyroid activity by administration of the drug thiouracil, and found that the amount of vitamin A stored in the liver was lessened, although the animals received adequate intakes of carotene, the vitamin precursor which is normally converted into the vitamin in the body. Possible explanations were that diminished thyroid activity impaired the conversion of carotene to vitamin A, or caused more rapid utilization of the vitamin. The correct explanation seems to have been provided by the study of Cama and Goodwin (34), who showed that thiouracil inhibited the absorption of carotene from the intestines, while the administration of dried thyroid substances increased the absorption.

In addition to pantothenic acid several other B vitamins have been implicated in endocrine functions. A deficiency of thiamine has been said to affect the ovaries leading to a cessation of sex cycles. Drill and Burrill (35) found this to be the case but the same effect was evident in animals which received thiamine but had the food intake restricted to the amount eaten by thiamine-deficient animals. Vitamin B_6 has been related to reproduction by Nelson and Evans (36); however, dietary deprivation of the vitamin was only slightly effective and it was necessary to add an anti-vitamin to the diet to obtain a pronounced change.

Since the pituitary is the "master" gland, in producing hormones which stimulate the activities of other endocrines, the effects of vitamin deficiencies on the pituitary have been given great attention. In 1937 Margitay-Becht and Wallner (37) reported that the growth effect of pituitary extract could not be obtained in vitamin A-deficient rats. Ershoff and Deuel (38) obtained a similar result. Beare and associates (39) observed a comparable lack of effect of the pituitary growth hormone in rats deprived of vitamin B_6, or of riboflavin, or of pantothenic acid. However, in all of these instances the criterion used to determine the effect of the growth hormone was whether it caused an increase in weight. In all cases the vitamin deficiency had produced a decrease in food intake and it was possible that insufficient food was available to permit a gain in weight. Beare et al. (39) reported that growth hormone had biochemical effects on vitamin B_6-deficient rats, although no growth was obtained. These observations illustrate the difficulty in interpreting studies in which observations are made on the effects of vitamin deficiencies on the endocrines. Animals deprived of a vitamin suffer from this deficiency and from inanition and both factors must be considered.

Effects of endocrine glands on nutritional requirements

Increased activity of the thyroid gland and an increased output of thyroid hormone augment the rate of energy production in the body. Consequently, there must be a greater food intake if body weight is to be maintained. This is perhaps the simplest example of various effects of endocrine activity on food needs. There are, however, other aspects to increased thyroid activity in addition to the need for calories. An increase in the production of thyroid hormone requires a greater amount of iodine. For years it has been assumed that an increase in the size of the thyroid in girls at the time of puberty is the result of the gland endeavoring to produce a needed greater quantity of hormone in the face of insufficient iodine; the classical work of Marine and Kimball (40) in 1921 in Akron provided the foundation for this explanation. There is considerable evidence that pregnancy stimulates the thyroid to secrete a needed, extra amount of hormone and one would expect that iodine requirement would be increased in pregnancy. Low-salt diets have proven to be useful in the prevention and treatment of the toxemias of pregnancy. For many persons in North America, and elsewhere, iodized salt is the main source of iodine. Obstetricians who have recommended the use of low-salt diets during pregnancy have not generally considered the question of probable iodine deficiency.

Several of the endocrines have marked effects upon protein utilization. The growth hormone of the pituitary gland depresses the production of energy from protein and causes an increase in the retention of protein in the body. One or more hormones of the adrenal glands have the opposite effect and bring about protein combustion. This latter effect is part of the "alarm reaction" to stress as described by Selye (41) in relation to the "general adaptation syndrome." While Selye's theory regarding stress has been made to fit almost every incident which might happen to humans, responses to certain types of injury are of interest in the present discussion. In 1933 Jones and Eaton (42) pointed out that severe protein deficiency often occurred in patients after certain types of operations and in 1936 Cuthbertson (43) described the protein wastage which occurs after bone fractures. Similar protein destruction in persons with severe burns was studied by Levenson and associates (44). Using Selye's alarm reaction as a basis, the increased combustion of protein after injury has been explained by Browne and Venning (45) as caused by an increased output of adrenal hormones. The production of the protein deficit after injury with consequent need for an increased intake has led to the use of high-protein supplements administered by injections, by tube-feeding, or in the diet if the patient is able to eat and swallow.

Several claims have been made that the growth hormone of the pituitary aggravates vitamin deficiencies and increases vitamin requirements. Ershoff and Deuel (38) found that administration of growth hormone to vitamin A-deprived rats caused precipitation of deficiency symptoms and accelerated death. Lotspeich (46) reported that supply of growth hormone aggravated the external signs of pantothenic acid deficiency. A similar observation regarding vitamin B_6 deficiency was made by Beare and associates (39). It is useful to point out that deficiencies of these three vitamins, whether due to the particular deficiency or to the concomitant inanition, all cause a decrease in body fat, an effect also produced by administration of growth hormone. This double effect on body fat may explain the aggravation of the deficiency state by growth hormone. This is especially true of vitamin B_6 with its well-known relation to fat metabolism.

Hormones from the endocrine glands may affect appetite, and hence, alter food consumption. Surgical removal of the pituitary gland causes a loss in appetite and food intake shrinks markedly. Administration of growth hormone to such an operated animal stimulates the appetite so that the animal eats considerably more food. It would be expected that a gain in body weight would result but Lee and Schaffer (47) recognized that this appetite effect should be considered in experimental work. They restricted the food supply of animals treated with growth hormone to the amount eaten by untreated control rats and showed that growth hormone had an effect on food utilization entirely separate from its effect on appetite. Another hormone which has a stimulating effect on appetite is insulin and this property of insulin has been used for some years to cause underweight persons to consume more food. Unfortunately, the effect of insulin on appetite has complicated the explanation advanced by Best and associates (48) that insulin acts as a growth hormone in bringing about protein retention and an increase in body weight. These investigators found that administration of insulin to rats from which the pituitary had been removed caused an increase in protein retention. They concluded that insulin could function as a growth hormone. However, the administered insulin caused an increase in food consumption and a similar result might have been obtained by forcibly feeding extra food without supplying insulin.

Summary

The primary factor deciding the nutritional state of an individual is the food intake. The nutritional state can be depreciated by impairments in digestion, in absorption, or in utilization of nutrients. The endocrine glands supply hormones which act as controllers of food utilization in the body and

various glands are themselves under the control of hormones produced by the pituitary. It would be expected that the activities of the endocrines would affect nutritional requirements. However, the endocrines, like all body tissues, are affected by supplies of nutrients. There are, then, two converse types of relations between nutrition and the endocrine glands.

A sustained and marked restriction in food intake has definite effects on the pituitary, altering first the output of hormones stimulating the glands of reproduction. The changes brought about by inanition are not only of importance in human nutrition but must be considered in interpreting laboratory research. In studies of single nutrient deficiencies part or all of the changes observed in the experimental animals may result from the inanition consequent to a loss of appetite, generally caused by dietary deficiencies.

Several nutrients have been specifically implicated in endocrine function. The adrenal glands contain a high concentration of ascorbic acid and the amount of this vitamin is rapidly decreased by administration of the adrenal-stimulating hormone of the pituitary. However, there is no clear evidence that a deficiency of ascorbic acid impairs the activity of the adrenal. Another vitamin, pantothenic acid, has been shown to be related to adrenal function and a deficiency of pantothenic acid does impair adrenal function.

At least some of the endocrine glands do influence nutrient requirements. Augmented activity of the thyroid increases the need for iodine and for calories. The pituitary growth hormone seems to increase requirements for several vitamins. In fact, the stimulating effect of growth hormone on appetite may be a natural method of obtaining larger amounts of vitamins and other nutrients. Increased activity of the adrenal, such as apparently takes place after stress or injury, promotes a greater combustion of protein and an increased intake of protein is indicated to preserve balance.

In the treatment of human disorders the interlocking influences of nutrition and the endocrine glands should be remembered. It is equally important that the interrelation be remembered in designing and in interpreting laboratory research, both by nutritionists and by endocrinologists.

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NEWS NOTE

In February, 1955 a three-day symposium was held at Michigan State College, East Lansing, Michigan on the theme "Nutrition of Plants, Animals and Man." The symposium, presented by the School of Agriculture, was one of a series of ten marking the college's centennial observation.

Borden's Review of Nutrition Research salutes Michigan State College on the attainment of this milestone and for the presentation of a most interesting symposium on a subject of considerable importance to nutritionists everywhere.

At one of the sessions, Dr. Josef Brozek of the Laboratory of Physiological Hygiene, School of Public Health, University of Minnesota, explored the link between nutrition and adult health.

The crucial causes of death in the United States; cardio-vascular disease, cancer and cerebro-vascular lesions, were considered in the light of nutrition. Dr. Brozek told his audience that there is not the slightest evidence that either general or specific nutritional deficiencies promote the development of diseases in this category.

Dr. Brozek concluded by citing further research in the field of "excess

nutrition" resulting in obesity, and extended hope for improving the situation by dietary means.

A report was presented at the symposium of a 10-year soil fertility research program now nearing completion by researchers at the Michigan Agricultural Experiment Station, and indicated that soil fertility does not change the composition or food value of crops materially. But it takes more than twice as much unfertilized land to feed an equal number of animals as can be maintained from crops grown on fertile soil.

The event brought more than 100 of the outstanding nutritionists in the nation to the Michigan State College campus for the first of 10 symposia to be held during the Centennial year of the nation's first college of agriculture.

Five Michigan State departments shared in the research and were assisted financially by the National Dairy Council and The Amercian Dairy Association.

Results showed:

Milk is the same — whether it comes from cows fed grain and forage from unfertilized soil or well-fertilized soil.

Reproduction of animals was not affected by soil fertility.

Animals fed forage from poor soil actually consumed slightly more and as a result produced slightly more milk. However, this difference was because fertilized forage, in many instances, was more stemmy and less palatable.

If a cow's ration is deficient in protein or minerals, milk production will decrease, but the composition of the milk will remain the same.